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POPULATION GENETIC STUDIES IN COD (*GADUS MORHUA* L.) BY MEANS OF THE HAEMOGLOBIN POLYMORPHISM; OBSERVATIONS IN A NORWEGIAN COASTAL POPULATION*

By

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ABSTRACT

MORK, J., GISKEØDEGÅRD, R., and SUNDNES, G. 1984. Population genetic studies in cod *(Gadus morhua* L.) by means of the haemoglobin polymorphism; observations in a Norwegian coastal population. *FiskDir. Skr. Ser. HavUnders., 17:* 449–471.

A total of 1205 immature and mature cod (*Gadus morhua* L.) of both sexes, caught during 1976–1981 in the Trondheimsfjord, Norway, were investigated in a population-genetic study by means of the polymorphic haemoglobin locus *Hbl* (SICK 1961). Genetic heterogeneity at this locus was observed both between and within samples, even when taken on the spawning ground. In 1977, 602 specimens from the spawning group were genotyped for *Hbl*, tagged with Lea hydrostatic tag, and released. The spatio-temporal distribution of genotypes in recaptures during four years reinforced the impression of genetic imbalance at *Hbl* for cod in this area. Much of the *Hbl* heterogeneity in adults was shown to be correlated with fish size, and an analyses of the length distribution among immatures revealed significant differences in mean lengths between the three common *Hbl* genotypes.

The goodness of fit, to our data, of two different population models is evaluated; in model 1, the observed *Hbl* heterogeneity and genotypic size differences are explained as effects of physical population mixing while in model 2, these observations are interpreted as effects of genotypic growth differences and size-selecting fishing gear.

The observations in the present study seemed most consistent under the latter model, although the former could not be formally excluded. The present findings are related to previous studies concerning the selection aspect of the cod *Hbl* polymorphism, and the necessity of experimental testing of some field observations is pointed out. However, it is concluded that there at present is reason to doubt the reliability of *Hbl* characteristics when used in cod population structure analyses.

* Contribution No. 215, Biological Station, Trondheim, Norway.

INTRODUCTION

The Atlantic cod (*Gadus morhua* L.) is thought to form several main stocks with respective distribution areas off eastern North America, at Newfoundland, Greenland, and Iceland, in the Barents sea, along the coast of Norway, in the North Sea, and in the Baltic Sea. Tagging experiments have shown a rather limited inter-stock exchange of individuals although the cod is an excellent swimmer, capable of long feeding- and spawning migrations.

In general, the results from applications of electrophoretic methods in cod population studies supported the traditional stock concept, but added some details to the picture. Thus, from studies by *e.g.* SICK (1965 a and b), FRYDENBERG, MØLLER, NAEVDAL, and SICK (1965), and CROSS and PAYNE (1978), further subdivisions into separate populations within main stocks were indicated for both Baltic cod, Norwegian coastal cod, and Western Atlantic cod.

The Trondheimsfjord cod, the object of the present study, is considered to be part of the Norwegian coastal cod stock which, according to FRYDENBERG *et al.* (1965), may consist of several populations, differing in their respective allele frequencies at the polymorphic haemoglobin locus *HbI* (*sensu* SICK 1961). The observed frequencies of HbI^1 displayed a cline along the coast of Norway; from 0.6–0.7 at the southern coast to 0.1 in the Barents Sea (FRYDENBERG *et al.* 1965).

A deficit of heterozygotes as compared to the expected values under genetic equilibrium (the WAHLUND effect) may indicate that a sample contains a mixture of individuals from two or more populations with different allele frequencies at the locus under study. In a pilot study in 1976, a statistically significant deficit of heterozygotes at *HbI* was observed among spawning cod in the Trondheimsfjord. However, the WAHLUND effect *per se* can give no information on the number of populations involved or their respective allele frequencies. Therefore, a combined conventional and genetic tagging experiment was designed in order to identify cod groups which possibly separate after spawning. If the observed genetic heterogeneity of the spawning group was an effect from population mixture, it might in this way be possible to sort out and characterize the individual groups by comparing allele frequences in groups of recaptures separated in time or space. Observations during the study seemed to call for explanations alternative to the assumption of population mixture as causes for heterogeneity at *HbI*. Accordingly, the aspect of natural selection was also considered in the subsequent experimental design and result evaluation.

MATERIALS AND METHODS

From the main cod spawning area in the inner part of the fjord (area 1 and 2 in Fig. 1) to the coastal outlet, the Trondheimsfjord is topographically divided into five main basins separated by relatively shallower tresholds (indicated by arrows in Fig. 1). These basins were in advance designated as separate recapture areas and numbered 1–5 in order of increasing distance from the release area. Coastal waters outside the fjord were accordingly designated as area 6.

During spawning, which occurs during March–June, ripe cod were caught in area 1 and 2 in 1976 and 1977. Immature cod were caught on two occasions; in the autumn 1977 and late autumn 1981. From 1977 and thereafter, length, age,

Figure 1. The Trondheimsfjord. Numbers 1–5 refer to the designation of separate recapture areas, which coincide with topographical basins separated by shallower thresholds (indicated by arrows). Tagged cod were released in area 1 and (partly) 2.

Table 1. Investigated samples. GN = gill net, BS = beach seine, SB = spinning bait.

Sample		Area	Gear	Sample size	Specimens scored for				Remarks
Code	Date				Sex	Length	Age	*HbI*	
A	Apr. -76	1;2	GN	100	0	0	0	100	Spawning cod
B	Mar.–May -77	1;2	GN	934	785	934	0	602	Spawning cod Tagging exp.
C	Oct. -77	3	BS	119	119	119	119	119	Immatures
D	Nov. -81	5	SB	41	41	41	41	41	Immatures

and sex data were recorded, when possible, for each *HbI*-genotyped specimen (Table 1).

All of the 934 cod in sample B (Table 1) were tagged (Lea hydrostatic tag) and released. From 602 of these, blood samples were drawn before release for the purpose of *HbI* genotyping. Sufficient amounts of blood could be drawn with a syringe from the thin veins inside the *operculum*. In samples A, D, and C, the blood samples were drawn from the heart of dead specimens. The blood was kept in tubes at 0–4° C during transport to the laboratory. Sample preparation and agar gel electrophoresis for haemoglobin genotyping were performed according to SICK (1961) within 12 hours. A total of 11 individuals with rare haemoglobin patterns (FRYDENBERG *et al.* 1965) was, for sake of tabular simplicity, excluded from the materials in the present report.

Using the same analytical procedures, the observed haemoglobin banding patterns appeared identical to those previously described (SICK 1961, FRYDENBERG *et al.* 1965). We therefore use the same locus and allele nomenclature as these authors. Age determination by otolith reading was performed according to ROLLEFSEN (1933).

The theoretical length at which 50% of a length group of fish will escape through the meshes of a net gear is by convention called the L_{50} of the gear. Samples A and B were obtained from professional fisherman who used gill nets with somewhat varying mesh size. However, the 119 immature specimens in sample C were caught with a small-mesh beach seine with L_{50} considerably below the length of the smallest 0-group cod caught.

STATISTICAL METHODS

Standardization procedures

In some cases, a non-parametrical statistical method, the KENDALLS τ-test (KENDALL 1962), was applied to the data. This made possible the test of trends in cases where the data did not lend themselves to objective grouping for the application of parametric tests, or where such tests failed to detect heteroge-

neity despite obvious trends in the material. In these cases, care was taken to fulfil, as near as possible, the underlying assuptions for this test. For instance, when allele frequencies were calculated from unequal sample sizes, they were standardized before application of the τ-test by means of the formula:

$$q_{s_i}' = \frac{q_{s_i} - q_p}{\sqrt{\frac{q_p\,(1-q_p)}{n_i}}} \qquad \text{I}$$

where

q_{s_i}' = standardized allele frequency in the i-*th* sample
q_{s_i} = observed allele frequency in the i-*th* sample
q_p = the true allele frequency in the population
n_i = number of genes in the i-*th* sample

Under the null hypothesis of no allele frequency differences between samples, q_s' approaches a normal distribution with expexted value = 0 and variance = 1.

Similarly, variables with continuous distributions, *e.g.* mean lengths, may be standardized by the formula:

$$U_i = \frac{\bar{X}_i - \mu}{\frac{\sigma_i}{\sqrt{n_i}}} \qquad \text{II}$$

where

U_i = standardized mean length in the i-*th* sample
$\bar{X}_i$ = calculated mean length in the i-*th* sample
μ = the true mean length (estimated from total group)
σ_i = standard deviation of mean length in the i-*th* sample
n_i = number of specimens in the i-*th* sample

Under the null hypothesis of no difference between sample mean lengths, U_1, U_2, U_3, ... U_n will be approximately normally distributed with expected mean value = 0 and variance = 1 if μ is known. Also, if μ is replaced by its estimated value, U_1, U_2, U_3, ... U_n will be approximately independent and approximately normally distributed with expected mean value = 0 and variance = 1.

Subgroup characterization by means of the WAHLUND *effect.*

If a sample contains a mixture of specimens from two populations with different allele frequencies at a certain polymorphic locus, the genotypic distribution at this locus will show a deficit of heterozygotes as compared to the expected equilibrium values calculated from the actual joint allele frequencies (the WAHLUND effect). The magnitude of this deficiency is a function of the true difference between subgroup allele frequencies and the proportion of individuals from each population. For instance; consider a population of size N with the frequency 0.4 for an allele *A*. A group of 0.5 N immigrants with q*A* = 0.5 will add N *A*-alleles to the gene pool, resulting in a joint q*A* = 0.433 and a heterozygote deficiency of size 0.0065 N. A joint q*A* = 0.433 may alternatively be obtained by the immigration of 0.058 N individuals with q*A* = 1.00. However, in the latter case the heterozygote deficiency after mixing will be of size 0.04 N, or numerically some six times higher than in the first situation.

If each of the involved population is in genetic equilibrium at the locus under study, and the allele frequencies in one of the populations (1) is known, the allele frequencies and proportion (in the mixed sample) of the other population (2) may be estimated by means of two sources of information, namely the change in observed allele frequency caused by the population mixing, and the size of the heterozygote deficiency in the mixed sample. This information is utilized in the following equiations:

$$pv + r(1-v) = a \qquad \text{III}$$

$$p^2v + r^2(1-v) = b \qquad \text{IV}$$

where

p = «known» allele frequency in population 1 (preferably estimated from a large sample)
r = unknown allele frequency in population 2
v = proportion of specimens from population 1 in the mixed sample
a = joint allele frequency (calculated from the mixed sample)
b = observed proportion of homozygotes for the actual allele in the mixed sample

The estimates of r and (1–v) are noted as $\hat{r}$ and $(\hat{1-v})$, respectively. If var (p) is sufficiently small to be neglected, the variances of $(\hat{r})$ and $(\hat{1-v})$ are estimated by the following expressions (derived by standard methods of first order approximation):

$$\text{var}(\hat{r}) \cong \frac{1}{[(1-v)(p-r)]^2} [(p+r)^2 \text{ var } (a) + \text{var } (b) - 2(p+r) \text{ cov } (a,b)] \qquad \text{V}$$

$$\text{var}\,(1\hat{-}v) \cong \frac{1}{(p-r)^4}\,[4r^2\,\text{Var}(a) + \text{Var}(b) - 4r\,\text{cov}(a,b)] \qquad \text{VI}$$

where

$$\text{var}\,(a) \cong \frac{1}{4N}\,[\beta(1-\beta) + 4\alpha(1-\alpha) - 4\alpha\beta]$$

$$\text{var}\,(b) \cong \frac{1}{N}\,[\alpha(1-\alpha)]$$

$$\text{cov}(a,b) \cong \frac{1}{2N}\,[-\alpha\beta + 2\alpha(1-\alpha)]$$

where

$\alpha = p^2v + r^2(1-v)$, *i.e.* $\alpha = b$
β = proportion of heterozygotes in the mixed sample
N = number of specimens in the mixed sample

The variances of $\hat{r}$ and $(1\hat{-}v)$ may be considerable. Extreme values of p, large sample sizes, large differences between p and r, and skew population proportions in the mixed sample will act to decrease the variances.

Sampling finite populations

Special problems may arise in connection with the application of statistical tests on samples from finite populations, *e.g.* the 591 specimens of cod (sample B) used in the genetic tagging experiment in this study. Allele frequencies, genotype proportions *etc* in the various recapture groups will not possess the status of independency which is a prerequisite for many test procedures, since the outcome in one sample may change the expectations for the next. When testing for heterogeneity among groups, this problem may be solved by including also the uncaptured group in the test, regarding this group as one of the samples.

RESULTS

By January 1981 27% of the genotyped specimens and 28% of the controls (not genotyped) in sample B had been recaptured. Significant additional mortality caused by the blood sampling was not found.

Most of the recaptures were reported from the Trondheimsfjord (*i.e.* area 1–5, Fig. 1). A few specimens, one genotyped, were recaptured in coastal waters north of the fjord. The most distant genotyped recapture was caught

NW of Andøya (Fig. 1). In the Trondheimsfjord the spatio-temporal distribution of recaptures seemed, not unexpectedly, to reflect an annual migration pattern correlated to the spawning-feeding cycle; in areas distant from the release area, recaptures were mainly performed in late summer and early autumn. Before each spawning season the concentration of recaptures moved towards the inner parts of the fjord.

A conventional analyses of variance did not reveal differences with respect to mean lengths of recaptures in the different areas (Table 2). There was, nevertheless, an obvious trend towards increasing mean lenght with distance from the release area towards the coastal outlet of the fjord (Table 3). Thus the specimens recaptured in the most distant areas were on average larger (at release) than those recaptured at shorter distances from the release area.

Table 2. Analyses of variance of mean lengths (at release) of recaptures in the various areas. Areas 5 and 6 are pooled.

H_0. The mean lengths (at release) are equal for the cod groups recaptured in the five areas.

$\alpha = 0.05$

Source of variation		Sum of squares	Degrees of freedom	Mean square
Total		5681.7358	158	
	Areas	122.7748	4	30.69
	Error	5558.9619	154	36.10

$F_{4,154} = 0.85$, $P = 0.496$
Therefore, do not reject H_0.

Table 3. Mean lengths (at release) of recaptures in areas 1–6 (increasing distance from the release area) during 1977–1981. The correlation between rank of standardized mean lengths and rank of recapture areas is tested by KENDALLS τ-test.

H_0. There is no correlation between rank of recapture areas and rank of mean lengths.
H_A: Rank of mean lengths is correlated to rank of recapture areas.

$\alpha = 0.05$

Recapture area	Number of recaptures	Mean length	Standardized mean length	Rank of standardized mean lengths
1	66	70.5	–2.44	1
2	31	71.9	–0.20	2
3	15	72.1	0.00	3
4	37	72.2	0.11	4
5	10	73.3	0.65	5

KENDALLS $\tau = 1$, $P = 0.0166$
Therefore, accept H_A.

HAEMOGLOBIN ANALYSES

The results from the electrophoretic analyses of haemoglobins are listed in Table 4. Although the four samples are taken from relatively nearby locations within the same fjord, there is a statistically significant heterogeneity in their genotypic compositions of haemoglobins. The main contributors to the calculated homogeneity chi-square of 13.158 are the marked heterozygote deficiency in sample A and the comparatively skew genotypic composition of sample D. If a chi-square homogeneity test of alleles, in stead of genotypes, is performed (2 × 4 contingency table), a chi-square value of 7.635 (d.f.=3) is obtained which is close to significance at the 5% level. In no individual sample, except A, did the proportion of genotypes deviate significantly from the expected equilibrium values calculated from individual sample allele frequencies. Sample C, which taken with nonselecting gear, showed a somewhat higher q*HbI*^{1} than the others, and also a weak excess of heterozygotes.

Table 4. Inter-sample homogeneity of *HbI* genotypic compositions tested by a $3 \times 4\chi^2$ contingency table (ecpected values under H_0 in parenthesis). The theoretical values calculated for intra-sample goodness of fit χ^2 tests (rightmost column) are not shown.

H_0: The genotypic compositions are homogeneous in the four samples
H_A: The genotypic compositions are heterogeneous in the four samples

$\alpha = 0.05$

Sample		Genotypes			N	q*HbI*^{1}	Goodness of fit χ^2 *)
Year	Code	*HbI*$^{2-2}$	*HbI*$^{1-2}$	*HbI*$^{1-1}$			
1976	A	35 (28.1)	39 (48.4)	26 (23.5)	100	0.455	4.568
1977	B	162 (166.0)	291 (286.1)	138 (138.9)	591	0.480	0.108
1977	C	24 (33.4)	65 (57.6)	30 (28.0)	119	0.525	1.060
1981	D	18 (11.5)	17 (19.9)	6 (9.6)	41	0.354	0.379
	A–D	239	412	200	851	0.477	0.751

*) If expected values are calculated from individual sample q*HbI*^{1}
$\chi^2_6 = 13.158$, $P = 0.0406$
Therefore, accept H_A.

Interestingly, the strongest indication of within-sample heterogeneity was observed in sample A; ripe cod caught in a very restricted area and period of time during spawning, *i.e.* when populations are ecpected to be negligibly mixed with each other.

In no individual sample, nor in pooled samples, was there any significant difference in allele frequencies between sexes.

Table 5. *HbI* genotypic composition and calculated allele frequencies in individual and pooled age groups of sample C. A χ^2 goodness of fit test is performed (pooled age groups, theoretical values in parenthesis).

H_0: The genotypes are distributed according to HARDY-WEINBERG ecpectations.

$\alpha = 0.05$

Age groups (years)	Number of genotypes			N	$qHbI^1$
	HbI^{2-2}	HbI^{1-2}	HbI^{1-1}		
0.5	1	7	4	12	0.625
1.5	2	2	1	5	0.400
2.5	13	31	17	61	0.533
3.5	8	25	8	41	0.500
0.5–3.5	24(26.8)	65(59.4)	30(32.8)	119	0.525

$\chi^2_1 = 1.060$, $P = 0.30$
Therefore, do not reject H_0.

Table 6. Mean lengths ($\bar{L}$) in *HbI* genotype groups in sample C. The standardized mean lengths are ranked in each group. The statistical significance of the observed ranking pattern is tested, assuming that the test statistic S from KENDALLS τ-tests within each age group is normally distributed with expected mean value = 0. When only one specimen was recorded in a genotype group, its length was ascribed an operational standard deviation (SD) equal to the arithmetic mean of the SDs of mean lengths in the two other genotype groups at that age in order to perform standardization.

H_0: $\bar{L}HbI^{2-2} = \bar{L}HbI^{1-2} = \bar{L}HbI^{1-1}$ in the observed age groups.
H_A: $\bar{L}HbI^{2-2} > \bar{L}HbI^{1-2} > \bar{L}HbI^{1-1}$ or $\bar{L}HbI^{2-2} < \bar{L}HbI^{1-2} < \bar{L}HbI^{1-1}$ in the observed age groups.

$\alpha = 0.05$

Age groups	Calculated, standardized (in parenthesis), and ranked (in italics) mean lengths (cm) of genotypes		
0.5	15.60 (1.13) *1*	12.90 (–0.09) *2*	12.43 (–0.37) *3*
1.5	30.25 (0.94) *1*	28.00 (–0.40) *2*	26.50 (–0.91) *3*
2.5	36.54 (0.99) *1*	35.21 (–0.41) *2*	35.03 (–0.55) *3*
3.5	43.94 (1.65) *1*	40.46 (–0.71) *2*	39.88 (–1.00) *3*

KENDALS τ-statistics: $S_{tot} = \Sigma S_{row} = 4 \times 3 = 12$. $Var(S_{tot.}) = \frac{[2n(n-1)(2n+5)]4}{36} = 14{,}67$

where n = 3; the number of groups in each ranking.
$S_{EXP.} = 0$. Employing the normal approximation of S gives $U = \frac{12-0}{\sqrt{14.67}} = 3.13$, $P = 0.0018$.

Therefore, accept H_A.

Hbl GENOTYPE AND FISH SIZE IN SAMPLE C (IMMATURES)

Taken with a small-mesh beach seine in a shallow area (depth < 2m), sample C consisted exclusively of immature specimens, 0.5 to 3.5 years of age (Table 5). The genotypic proportions and allele frequencies in age groups showed no significant deviations from the expected equilibrium values. However, a very special pattern appeared when mean lengths of genotypes within each age group were calculated: In each of the four age groups, the HbI^{2-2} homozygotes displayed the highest and the HbI^{1-1} homozygotes the lowest mean lengths, with heterozygotes intermediate in all cases (Table 6). The probability of obtaining such a pattern merely by chance is very low, as indicated by the employed test (Table 6). The 3.5 year age group (Table 5 and 6) probably contained a considerable proportion of individuals, particularly among the males, which would have recruited to the spawning stock the following year. The lenght difference in favour of the HbI^{2-2} compared to the HbI^{1-1} genotype in this age group was about 10%, corresponding roughly to a weight difference of 34% (assuming a condition factor of 1). The egg number in cod is a linear function of body weight (Daan 1975). Also, better growth may cause earlier maturation. Potentially, both effects will, if allowed to act unmodified, increase the proportion of the HbI^2 allele in each new generation.

Hbl GENOTYPE AND FISH SIZE IN SAMPLE B (SPAWNING COD)

In general, otoliths were not returned together with the tags in recaptures from sample B. Thus the specimens in this sample could not be aged. However, from an expected positive correlation between fish size and age, information on a potential variation in haemoglobin allele frequencies with age may be obtained by the comparison of qHbI^1 in different size groups. Because of the difficulties connected with an objective size grouping for such purposes, it was decided instead to test for trends, using Kendalls τ-test of rank correlation. The size range of the specimens in sample B was 55–108 cm. When observed lengths were grouped in 1.5 cm intervals (*i.e.* 55–56.5, 57–58.5, . . . 107–108.5), 21 intervals were occupied. The qHbI^1 among individuals within each interval was calculated and then standardized according to I (cf Materials and Methods). As revealed by Kendalls τ, the hereby obtained positive correlation between increments in length and increments in standardized qHbI^1 was statistically significant at the 1% level (Kendalls τ-statistics; S=84, n=21. Employing the normal approximation reveals U = 2.56, P = 0.01). Thus we may accept with some confidence that the HbI^1 allele was more frequent among larger (and presumably older) individuals in the spawning stock in 1977.

RESULTS FROM THE GENETIC TAGGING EXPERIMENT

Genotypic distribution of recaptures in time

In Table 7, the recaptures from sample B are arranged in groups according to year of recapture. As shown, a chi-square analyses did not reveal heterogeneity in the annual distribution of genotypes. Nor does a chi-square contingency table of alleles instead of genotypes show significant heterogeneity between years (2×5 contingency table, $\chi^2_4 = 6.21$, $P = 0.184$). A considerable proportion of the recaptures in 1977 was taken in the release area during the tagging period, and as shown in Table 7, the recaptures for this year are rather similar to the released group in allele frequencies and genotypic proportions. Among recaptures in later years there is, in Table 7, a weak trend towords a higher proportion of the Hbl^2 allele early in the period. The number of groups in Table 7 is too low for the application of trend tests. However, if recapture groupings in half-year intervals is performed (excluding first half of 1977 for the reasons mentioned above) seven groups are obtained. From the last half of 1977 and thereafter, the calculated and standardized qHbl^1 in these groups form the series: -0.4, -1.8, -0.9, 0.2, 0.9, 0.8 and 0.9. As revealed by a KENDALLS τ-test this is a significant correlation between time and increasing qHbl^1 ($\tau = 0.667$, $P = 0.036$), which presumably may be interpreted in terms of higher survival rates for Hbl^1-possessing individuals.

Table 7. Observed and expected (in parenthesis) genotypic distribution and calculated allele frequencies at *Hbl* in recapture groups and the not recaptured group from sample B. Expected numbers are derived from the genotypic proportions in sample B. The homogeneity of the genotypic compositions of recapture groups is tested ($3 \times 4\ \chi^2$ contingency table; years 1979 and 1980 are pooled and the not recaptured group is included in the test for theoretical statistical reasons).

H_0: The genotypic compositions are homogeneous in the four groups.

$\alpha = 0.05$

Year of recapture	Number of genotypes			N	qHbl^1
	Hbl^{2-2}	Hbl^{1-2}	Hbl^{1-1}		
1977	27 (26.3)	43 (47.3)	26 (22.4)	96	0.495
1978	17 (11.5)	19 (20.7)	6 (9.8)	42	0.367
1979	3 (3.9)	7 (6.8)	4 (3.3)	14	0.536
1980	0 (1.9)	5 (3.5)	2 (1.6)	7	0.643
Sum recaptures ..	47 (43.6)	74 (78.3)	38 (37.1)	159	0.472
Not recaptured ..	115 (118.4)	217 (212.7)	100 (100.9)	432	0.483
Total (= sample B)	162	291	138	591	0.480

$\chi^2_6 = 7.303$, $P = 0.293$

Therefore, do not reject H_0.

Genotypic distribution of recaptures in space

The calculated allele frequencies at *HbI* among recaptures in the six recapture areas form a highly noticeable pattern; from area 1 (the release area) towards area 6 (coastal waters) there is a steady decrease in qHbI^1 in recapture groups (Fig. 1 and Table 8). As shown in Table 8, the rank of the allele frequencies is not altered by the standardization procedure, and the observed trend is highly significant, although conventional chi-square analyses failed to detect genetic heterogeneity among groups from the different areas: With the not-recaptured group of 432 specimens included and groups from areas 5 and 6 pooled, a 3×6 chi-square contingency table revealed $\chi^2_{10} = 7.908$, P = 0.638. When testing for allele frequency differences (2 × 7 contingency table, no pooling), $\chi^2_6 = 5.356$, P = 0.499. However, from the significant trend, which is observed (Table 8), it may be concluded that the various *HbI* genotypes among tagged spawning cod in 1977 formed a highly specific, nonrandom spatial distribution pattern in subsequent recaptures within the fjord. This trend was observed already in the 1977 recaptures and has been only slightly reinforced later. Table 8 also shows that the recaptures in area 5 have deviating qHbI^1 at the same level as that of immature specimens from this area (sample D, Table 4).

Table 8. Observed and expected (in parenthesis, derived from genotypic proportions in sample B) genotypic distribution and calculated and standardized allele frequencies in recaptures from different areas. Recapture areas are ranked 1–6 in order of increasing distance from the release area towards coastal areas. Standardized allele frequencies are ranked 1–6 from highest to lowest value. The significance of the correlation between these two rankings is tested by KENDALLS τ-test.

H_0: The rank of recapture areas and rank of standardized allele frequencies are independant.
H_A: There is a correlation between rank of recapture areas and rank of standardized allele frequencies.

$\alpha = 0.05$

Area of recapture	Number of genotypes			N	qHbI^1	Standardized qHbI^1	Rank of standardized qHbI^1
	HbI^{2-2}	HbI^{1-2}	HbI^{1-1}				
1	17 (18.1)	30 (32.5)	19 (15.4)	66	0.515	0.805	1
2	9 (8.5)	13 (15.3)	9 (7.2)	31	0.500	0.315	2
3	3 (4.1)	10 (7.4)	2 (3.5)	15	0.467	–0.143	3
4	13 (10.1)	17 (18.2)	7 (8.6)	37	0.419	–1.050	4
5	4 (2.5)	4 (4.4)	1 (2.1)	9	0.333	–1.248	5
6	1 (0.3)	0 (0.5)	0 (0.2)	1	0.000	–1.358	6

KENDALLS $\tau = 1$ (6 pairs), P = 0.0028 (two-sided)
Therefore, accept H_A.

HbI genotype and fish size in recaptures from sample B

The trend of increasing qHbI^1 with increasing fish size among the releases in the tagging experiment was also observed among the 159 recaptures. However, from the qHbI^1s in the 1.5 cm lenght intervals the change in allele frequencies seemed less smooth among the recaptures. Rather, a kind of shift appeared at about 73 cm length. This coincided with a marked gap in the length-frequency curve for the releases. Thus, among recaptures, the qHbI^1s in the 1.5 cm intervals < 73 cm ranged from 0.25–0.46 (joint allele frequency for HbI^1 = 0.40 in 100 specimens) while the length groups > 73.5 cm showed qHbI^1 values ranging from 0.50–0.75 (joint qHbI^1 = 0.59 among 59 specimens). The genetic composition in these two groups appeared with insignificant deficit and excess of heterozygotes, respectively, among smaller and larger cod. Although testing of differences between groupings based on experience may be a statistically hazardeous procedure, it is noted that the observed allele frequency difference (0.4 and 0.59) reveals $\chi^2_1 = 11.06$, P = 0.0009.

HbI GENOTYPIC DISTRIBUTION IN AGE GROUPS IN SAMPLE D (IMMATURES)

Four age groups (1, 2, 3, and 4 years of age) were represented in sample D. The genotypic mean lengths within age groups appeared to be without any significant trend (cf sample C), but there were somewhat smaller age-group mean length differences than in sample C, which may indicate that the length distribution of specimens in this sample had been affected by the fishing gear (spinning bait, which may have a size-dependant attraction for individuals of the same age group). The mean lengths in succeding age groups (1–4 years, number of specimens in each age group in parenthesis) were; 30.0 (1), 31.5 (17), 35.4 (22), and 46 (1). For the purpose of comparing *HbI* allele frequencies the material was pooled as two groups; 1 + 2 year and 3 + 4 year. Interestingly, the HbI^2 allele was much better represented in the former group, the individuals of which had presumably been selected among the larger 1 and 2 year old cod (qHbI^1 = 0.19 (N = 18) and qHbI^1 = 0.48 (N = 23) for younger and older cod, respectively. χ^2_1 for the difference is 7.04, P = 0.008).

LENGTH AT RELEASE AND PROBABILITY OF RECAPTURE

Analyses of mean lengths of recaptures in 1977 compared with the rest of the period (1978–1980) revealed a general trend that large size at release increased the probability of recapture (Table 9). It seems reasonable to relate this trend to the size-selecting effect of the fishing gear used in these areas. However, under such a catch regime it is surprising that the HbI^2-possessing specimens, which on average were smaller at release, are overrepresentated in recaptures

Table 9. Mean lengths (at release) of specimens (numbers in parenthesis) from sample B which were recaptured in 1977 versus the period 1978–1980. Note the consistently higher mean lengths for the 1977 recaptures in all the recapture areas.

Area	Mean length at release for recaptures in	
	1977	1978–1980
1	70.69 (39)	70.53 (27)
2	75.05 (19)	71.70 (12)
3	73.67 (9)	69.67 (6)
4	72.30 (24)	71.20 (13)
5	75.00 (5)	71.50 (5)

taken early in the period 1977–1980. For instance, the 1978 recaptures, where the HbI^2-allele was very frequent (Table 7), consisted of specimens whose mean length at release was lower than that for those recaptured in the rest of the period (1979–1980). This pattern applied to all the five recapture areas in the fjord and was thus statistically significant (Sign test, $P = 0.03$).

A possible explanation of these findings is higher growth rates for the HbI^2-possessing individuals subsequent to release. Unfortunately, due to a general lack of reliable information on fish lengths in recapture reports, these potential genotypic growth differences could not be controlled directly.

DISCUSSION

Significant indications of some form of genetic imbalance at *HbI* were observed in all samples. These and other observations may be listed as follows:

1. Almost all (99%) the recaptures during 4 years were taken in the Trondheimsfjord.
2. The mean length (at release) in recapture groups increased with distances from the release area towards coastal waters.
3. Genotypic heterogeneity at *HbI* was oserved between four samples taken within the same fjord. A potential WAHLUND effect was statistically significant in one sample (A) which was taken from the spawning group in 1976.
4. In gill net catches of spawning cod (sample B) the HbI^2 allele was over-represented among smaller (and presumably younger) cod, and there was a trend of increasing $qHbI^1$ with fish size (and presumably age).
5. Generally, large size at release was positively correlated with recapture early in the period 1977–1980, but there were deviations from this rule which indicated genotype-related growth differences, favouring HbI^2-possessing individuals, subsequent to release.

6. Despite observations 4 and 5, HbI^2-possessing specimens were over-represented in early recaptures and under-represented in late recaptures, resulting in increasing qHbI^1 with time in recaptures from sample B during 1977–1980.
7. Despite observations 2 and 4, the qHbI^1 among recaptures decreased with distance from the release area towards coastal waters.
8. In samples of immature specimens, qHbI^1 was lower in coastal areas than in the inner parts of the fjord.
9. Size differences related to haemoglobin genotype were observed among immature individuals. In sample C, the rank of mean lengths among genotypes was $HbI^{2-2} > HbI^{1-2} > HbI^{1-1}$ in four succeeding age groups (0.5–3.5 years of age).
10. Significant age group qHbI^1 differences were found in sample D (immatures).

In the following attempts to fit models to our observations it is assumed that these are valid for normal occurrences and permit generalizations about the biology of cod in this area.

It must first be stated that A, B, C, and D are very unlikely to be random samples from one and the same population, statistically ideal with respect to *HbI*. Thus the remaining possibilities which will be evaluted are a multiple population model (represented by the simplest form; two populations), and an alternative model which postulates deviations from statistical ideality at *HbI*. The latter is referred to as the selection model.

THE TWO-POPULATION MODEL

Under this model the qHbI^1 cline with distance from the release area in recaptures from sample B (Table 8) indicates differences in the migration behaviour of the populations involved. Thus, in the following a distinction is made between a migrant population with low qHbI^1 and a local population with higher qHbI^1.

If samples A, B, C, and D, all of which show some form of genetic heterogeneity, were mixtures of different proportions of specimens from migrant and local cod, virtually all the observations listed in pts 1–10 (above) could be explained by making certain assumptions. These assumptions are; a very effective reproductive isolation (cf obs. 3), a relatively large difference in qHbI^1 between populations (cf obs. 3, 7, 8, and 10), population differences in fish size at the same age (migrants being the largest, cf obs. 2, 7, and 9), and differences in total annual mortality (or some other form of catch unavailability which is highest for migrants, cf obs. 4 and 6).

Since migrant and local cod occur simultaneously in the same area during spawning, the actual isolation mechanisms must probably involve ethological barrieres. However, although bimodality of catch rate curve, or pelagic egg density has not been observed (unpublished data), it is possible that the very

long spawning period (4 months) in this area may consist of two overlapping spawnings. Potentially, such a spawning separation would contribute to the reproductive isolation, and if the migrant cod on average release their sex products somewhat earlier in the season, the offspring would presumably get an initial growth advantage which could explain the observed genotypic mean length differences in mixed samples (cf obs. 9). Also, such a spawning pattern, in combination with effects from the estuarine circulation in the Trondheimsfjord, could explain the observation of lower qHbI^1 among immatures in the coast-near areas (cf obs. 8). The lower water temperatures early in the spawning season will depress the development rates of eggs and larvae and thereby prolong the pelagic stage. A prolonged pelagic stage will tend to increase the distance which they are carried with the surface currents towards the coastal outlet of the fjord before they settle. The significant qHbI^1 difference between young and older immature specimens in sample D (cf Results) might indicate either annual variation in the separating effect of such current transport, or alternatively that the effect is leveled out by subsequent mixing of individuals from both populations throughout the fjord (*i.e.* in sample D age groups 1 and 2 are pure migrants, while 3 and 4 are mixed).

None of the samples A, B, C, and D provides estimates of the haemoglobin allele frequencies in unmixed populations, but the younger specimens in sample D might be a sufficiently homogeneous group as to indicate the qHbI^1 among migrants (qHbI^1 = 0.194 (N = 18) in age group 1 and 2, cf Results).

Under this assumption, the application of formulas III and IV (cf Materials and Methods) gives the following results, in terms of *HbI* allele frequencies and population proportions necessary to create the observed genotypic distributions in sample A, B, and D:

Sample	Local cod proportion	Local cod qHbI^1
A	0.56 (S.E. = 0.13)	0.66 (S.E. = 0.09)
B	0.96 (S.E. = 0.10)	0.49 (S.E. = 0.04)
D	0.55 (S.E. = 0.45)	0.48 (S.E. = 0.22)

These qHbI^1 estimates are rather different, but their heterogeneity does not exceed the 5% probability level ($P>0.06$). Thus, although it is not very probable, the consistency of the observed genotypic distributions in samples A, B, and D under the two-population model cannot be formally excluded.

THE SELECTION MODEL

Frequently, in the present materials, indications of heterogeneity at *HbI* were connected with fish size differences and the use of size-selecting gear. In the two-population model above, this connection was explained as population-

mixture effects. It might, however, point to intra-population *HbI* genotypic size differences. The latter explanation permits the construction of a population model without assumptions of population co-existence and reproductive barriers.

Under this model, the reason for the various observations of genetic imbalance is deduces from the observations in our only unselected sample with respect to fish size; sample C. The highly significant rank of genotypic mean lengths in that sample (Table 5) may seem to be an unexpectedly dramatic manifestation of adaptional differences for commonly occuring alleles at one

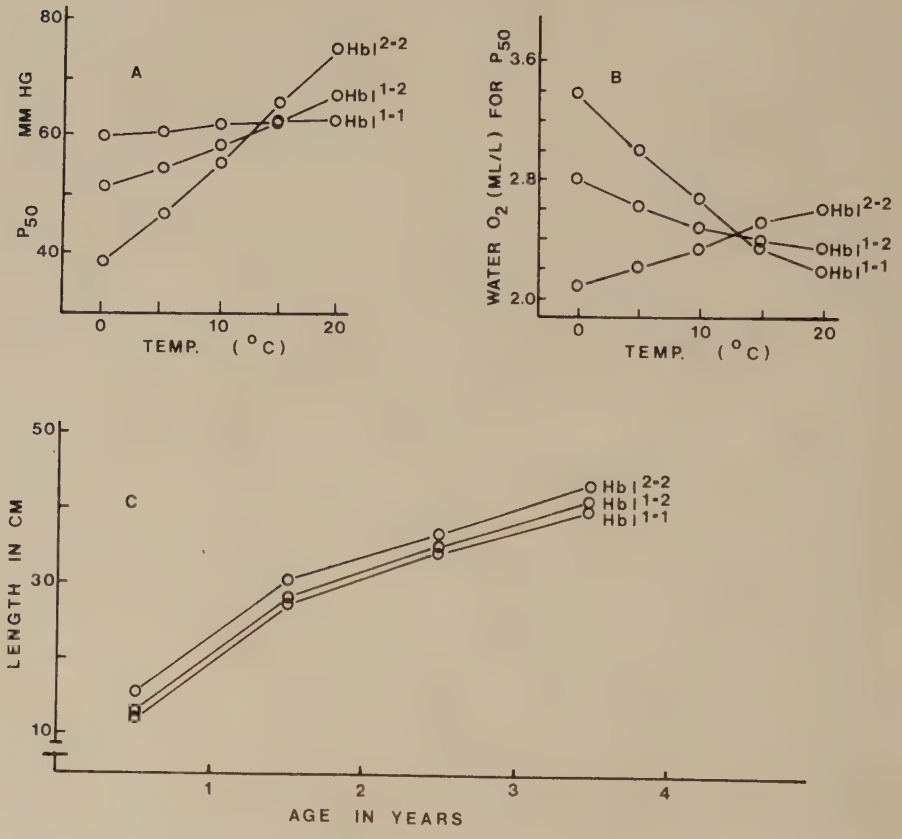

Figure 2. A) Necessary oxygen partial pressure at various temperatures for half-saturation of cod erythrocytes. Note the different effect of temperature for the three most common *HbI* genotypes. B) Necessary oxygen concentration (ml/l) in sea water for half-saturation of cod erythrocytes, further emphasizing the genotypic differences in functional properties related to temperature regimes.

C) Calculated mean lengths of *HbI* genotypes in four age groups of immature cod in sample C. Note the consistant ranking of genotypes. A) and B) are redrawn from Karpov and Novikov (1980).

locus, although haemoglobins no doubt are significant proteins in the interaction between organisms and environment. However, the findings of Karpov and Novikov (1980), in a study on the functional properties of the three common cod haemoglobins in relation to temperature, provide a physiological basis for the observed size differences: These authors demonstrated experimentally that at low temperatures the HbI^{2-2} molecule was by far the most efficient oxygen carrier, while the HbI^{1-1} type showed similar advantages at high temperatures. The heterozygous type HbI^{1-2} was consistently intermediate in performance (Fig. 2 A and B).

As indicated by the time for deposition of opaque otolith material, the main growth period for the immature specimens in sample C was autumn and winter, *i.e.* when the water temperature regime in these areas is expected to favour the HbI^2-possessing specimens. (The «neutral» temperature point indicated by the curves in Fig. 2 A and B is approximately 13° C. In fact, at 5 m depth in the Trondheimsfjord this temperature is not exceeded even in the warm summer months, and at 2 m depth only in July (referring to mean values for 1963–1975)). Thus the genotypic mean lengths in sample C (Fig. 2 C) vary in the expected manner based on molecular and growth period knowledge. However, there are alternative explanations: If the possession of an effective respiratory agent is advantageous for general body growth, it may, potentially, also be capable of affecting the gonadal maturing process within each spawning season. If so, and if the HbI^2-possessing specimens on average release their sex products somewhat earlier, the offspring might get an initial growth advantage which, if subsequently maintained, would be manifested as shown in Fig. 2 C. As outlined in the two-population model (above) such a spawning pattern might also, by the estuarine transport effect, explain the lower qHbI^1 in areas distant from the spawning grounds (*e.g.* area 5, cf Table 4). On the other hand; under the present model the low qHbI^1 in sample D is perhaps equally well explained as an artifact caused by the use of selecting gear; only the largest individuals in age group 1 and 2 may have been attracted and caught by the spinning bait used (cf qHbI^1 age group differences in sample D; Results.)

Similarly, the *HbI* genotypic heterogeneity in sample A (Table 4) may be explained as a joint effect of genotypic size differences and size-selecting gear. Potentially, moreover, better growth rates might cause maturation at lower age, which would contribute to a lowered qHbI^1 and heterozygote deficiency in samples from the spawning part of the population. From sample B one may get an impression of the dynamics of such an artificial selection pattern: The over-representation of the HbI^2 allele among the smaller fish (which were only just retained by the meshes) accords to the expectations. The potential qHbI^1-changing effect of this selection may be considerable. We have estimated that among adult Trondheimsfjord cod, more than 60% of the total annual mortality is contributed by (size-selecting) net gear (unpublished data). Since a

size selection probably will affect a given yearclass of cod for several succeeding years, one would expect that this unproportional removal of HbI^2-possessing specimens would gradually reduce the frequency of this allele as the cod grow older. In fact, the trend of increasing qHbI^1 with size (and presumably age) in sample B (cf Results) indicates just this effect. In addition, the increasing qHbI^1 with time during 4 years in recaptures from sample B (cf Results) supports the existence of such a selection regime. Finally, the deviation, for HbI^2-possessing specimens, from the overall pattern that low length at release reduces the probability of an early recapture (cf Results and obs. 5 above), accords to the expectations if higher growth rates is the reason for HbI^2-possessing specimens being more effectively removed from the population during the recapture period.

Actually, the outlined selection pattern might be a crucial part of the present model; the qHbI^1-lowering effect, in succeeding generations, of the larger size (and thereby higher fecundity) of the HbI^2-possessing specimens is effectively counteracted by their unproportional removal before spawning, and lower representation in older age groups. Otherwise it might be difficult to explain the apparent stability of qHbI^1 for cod in the Trondheimsfjord; the joint q$HbI^1 = 0.477$ in the present study is close to the value reported by Frydenberg *et al.* (1965) for cod in this area (0.485 based on 163 specimens caught in 1963).

The observation that the mean lengths (at release) of recaptures from sample B increased with distance from the release area (Table 3) may reflect a general tendency of large cod specimens to perform longer migrations in the spawning intervals, irrespectively of *HbI* traits. If so, the trend of decreasing qHbI^1 among recaptures with distance from the release area (Table 8 and obs. 7 above) is not easily explained without postulating some form of inter-spawning locality confinement; *e.g.* that adult specimens tend to return to their former nursery grounds after each spawning, and that the qHbI^1 on these nursery grounds declines towards the coastal outlet of the fjord due to the estuarine transport effect outlined above. However, if the trend of increasing mean lengths among recaptures with distance from the release area is an artifact caused by different gear being used in the different areas (in 1977, the percentages of gill-net caught recaptures in areas 1–5 were approximately 100, 100, 25, 10, and 0, with lure and longlines as alternative gears), then the observed qHbI^1 cline may also be an expected effect from rapid growth of HbI^2-possessing specimens.

CHOICE OF MODEL

One of the strict underlying assumptions for the two-population model was a complete reproductive isolation, presumably administered by ethological barrieres. Although such barrieres previously have been suggested to exist between cod populations (Möller 1968 and 1969 in studies of Arcto-

Norwegian cod and Norwegian coastal cod in Lofoten and the fjords of Northern Norway (Fig. 1), they have not been demonstrated experimentally and their existence remains uncertain. Also, one might empirically expect the co-existance of two separate populations within the same fjord to rely on the utilization of different ecological niches. However, under the two-population model, the present results indicate that mixing occurs at all age stages among immatures (cf sample C) and throughout the fjord (cf sample D). These objections, together with the relatively (but not significantly) inconsistant genotypic proportions observed in the various samples (cf text above), reduce our confidence in a two-population hypothesis.

The selection model seems to fit better to our observations. Although some of the suggested explanations under this model may at present be speculative and may require later adjustments, the evidence of genotypic size differences and fishing gear selection of genotypes appeared consistent. However, since the two-population model could not be formally excluded, the present indications of *HbI* genotypic growth rate differences are all indirect and should preferably be tested experimentally.

The seemingly stable qHbI^1 between 1963 and 1977–80 (cf text above) is not incompatible with this model; it might be expected that the system has had time to reach a (pseudo-)equilibrium where the different forces involved cancel out each other. Although not considered above, the possibility of higher survival rates of HbI^1-possessing specimens under certain conditions (*e.g.* warm summer habitats) can not be excluded. Potentially, such differential genotypic mortality would act together with size-selecting gear to counteract the qHbI^1-lowering effect of larger size of HbI^2-possessing cod females, but would probably not provide long-term stability of *HbI* allele frequencies. A potential stability would seem to require the action of other agents, such as *e.g.* a stable supply of HbI^1 genes from other populations, or an overall (fecundity plus mortality) heterozygote advantage at *HbI* in the Trondheimsfjord. These, and other possibilities, in the potentially complicated dynamics of this polymorphism can only be evaluated through further investigations.

The suggestion that selection forces of detectable magnitude act on the *HbI* system is not new; this possibility has been considered by several authors (Sick 1965 a, Frydenberg *et al.* 1965, Frydenberg, Nielsen and Sick 1967, Frydenberg, Nielsen and Simonsen 1969, Wilkins 1969, Karpov and Novikov 1980, and Kirpichnikov 1981). In a recent study, Mork *et al.* (1982) reported observations on sexual differences in qHbI^1 among cod from trawl catches in the Trondheimsfjord. Sex-related qHbI^1 differences were also reported by Frydenberg *et al.* (1969) for cod from Danish waters. Clearly, if a selection is working, such effects can not be excluded. However, as discussed above, it is difficult to determine whether these findings reflect reality or are artifacts caused by the sampling with size-selecting gear; the faster growth of female cod compared to males may, dependant upon the sampling gear, cause

an unproportional representation of the sexes from various age groups in the sample and through effects such as those discussed above induce false estimates of allele frequencies in the sexes. Hence, it appears that cod samples which are representative of the population as a whole with respect to *HbI* are not easily obtained under the present model.

As mentioned above there is, under the selection model, room for discussion of the causal relationships. In the above discussion we have not considered the possibility that the experience of the stress or discomfort connected with certain temperature regimes may cause a spatial segregation of genotypes. Potentially, such a segregation could be manifested as genetic heterogeneity in samples and also affect growth rates, catch rates, mortality rates and the spatial/temporal distribution of genotypes. In fact, Karpov and Novikov (1980) reported observations that individual cod actively chosed habitats with temperature regimes suitable for their *HbI* genotype. Those observations were not adequately documented, but are nevertheless very interesting and may be tested experimentally. If confirmed, the ability of active habitat selection seems compatible with the present selection model and may provide alternative explanations to some of the cases of genetic heterogeneity at *HbI* reported above.

Our choice of the selection model as the better explanation of the present observations does not necessarily challenge the conclusions from earlier studies on cod stock and population structure based on *HbI* analyses, which in fact agreed well with results from conventional approaches (cf, *e.g.*, Möller 1968 and 1969, Sick 1965 a and b *versus* Schmidt 1930). However, there seems now to be sufficient evidence, from several independent studies, to question the reliability of characteristics at *HbI* for use in population structure analyses, particularly when allele frequency differences are on the fine scale.

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